A STUDY OF SURGICAL WOUND INFECTION IN GOVERNMENT RAJAJI HOSPITAL, MADURAI

DISSERTATION SUBMITTED FOR

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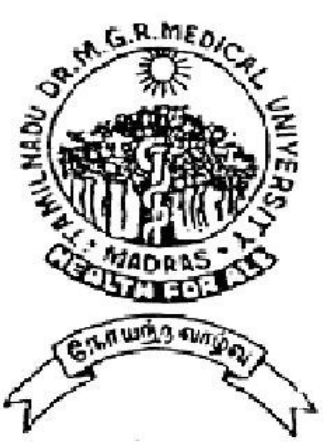

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY CHENNAI

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY OF SURGICAL WOUND INFECTION IN GOVT. RAJAJI HOSPITAL, MADURAI"** submitted by **Dr. R. KANNAKI** to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S Degree Branch –I (General Surgery) is a bonafide research work were carried out by her under direct supervision & guidance.

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DECLARATION

I **Dr. R. KANNAKI** declare that, I carried out this work on, **"A STUDY OF SURGICAL WOUND INFECTION IN GOVT. RAJAJI HOSPITAL, MADURAI"** at the Department of Surgery, Govt. Rajaji Hospital during the period of June 2006 to June 2008. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any other University, Board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery.

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CONTENTS

INTRODUCTION

Hundreds of Millions of People around the world undergo surgery each year. Infection of the surgical site (formerly referred to as 'wound infection', terminology that is no longer used owing to confusion between infections of surgical incisions and those of traumatic wounds) is a consequence of surgery, but it is not inevitable. Surgical site infection (SSI) entirely the fault of the operating surgeon, despite the contrary belief of many members of the quality improvement bureaucracy. This review will examine the factors that increase the risk of surgical site infection, and interventions that can decrease the incidence of infection.

Surgical site infection is most challenging to every surgeons and each and every body is trying their methods to reduce the problem.

Before the mid-19th century, surgical patients commonly developed post operative 'incapacitating fever', followed by purulent discharge at the site of surgical incision followed by overwhelming sepsis, and often death would result. It was not until the late 1860s, after Joseph Lister introduced the principles of antisepsis, the postoperative infection associated morbidity decreased substantially. There was a radical change in surgery after Lister's

pioneering work which resulted in shift in the surgical outcome from infection, high morbidity and mortality to a procedure that could eliminate suffering and prolong life.

The centers for Disease Control and Prevention (CDC) and National Nosocomial Infections Surveillance (NNIS) system, established 1970, monitors reported trends in nosocomial infections in US acute care hospitals. Based on NNIS system reports, Surgical Site Infections (SSIs) are the third most frequently reported nosocomial infection, accounting for 14 to 16 percent of all nosocomial infections among hospitalized patients.

Among surgical patients, surgical site infections were the most common nosocomial infection, accounting for 38 percent of all such infections. Of these surgical site infections two third were confined to the incision, and one third involved organs or spaces accessed during surgery. When surgical patients with nosocomial SSI died, 77 percent of the deaths were due to infection and the majority (93%) were serious infections involving organs or spaces accessed during the operation.

Advances in infection control practices include improved operating room ventilation, sterilization methods, barriers, surgical technique, and

availability of antimicrobial prophylaxis. Despite these activities, SSIs remain a substantial cause of morbidity and mortality among hospitalized patients. There also are increased numbers of prosthetic implant and organ transplant operations performed. Thus, to reduce the risk of surgical site infection, a systemic but realistic approach must be applied with the awareness that this risk is influenced by characteristics of the patient, operation, personnel and hospital. Paramount significance need be given to all the equipment that is used on the patient rather than the inanimate environment of air quality. This implies that monitoring of Central sterile service departments (CSSD) is a necessity for the success of a procedure.

AIM OF STUDY

- Surgical site infection is most challenging to every surgeons and each and every one is trying their own methods to reduce the problem
- So our aim is also to try new modalities to reduce the surgical site infection

MATERIALS AND METHODS

- We tried 4% chlorhexidine gluconate for topical antiseptic at site instead of povidone Iodine.
- Using Hair clippers instead of pre operative saving
- Insisting the use of cap and mask by all medical and paramedical personals inside the operation theatre.

Method :

The patients are instructed to take bath with ordinary antiseptic soap in the previous evening and the morning on the day of surgery.

At the operation site the skin is prepared immediately before surgery by removing the hair by clipping and thorough washing done with antiseptic soap.

On the table before drapping the operation site painted with 4% chlorhexidine antiseptic solution and allowed to dry.

All personals inside the theatre are instructed strictly to wear cap and mask and separate theatre dress.

Microbiology swab sent for every infected cases for culture and sensitivity.

TERMINOLOGY AND DEFINITIONS OF SURGICAL SITE INFECTION

What constitutes a Surgical site infection ? There is little agreement on the matter with respect to the appearance of the incision, even among experts. Is it cellulites of the incision without drainage or, alternatively, non-purulent drainage without cellulites? Is any incision infected that must be reopened, or is the requirement for antimicrobial therapy the best indicator? Most experts agree that surgical sites that do not harbor purulent fluid are not infected, but the lack of agreement otherwise means that any retrospective study of surgical site infection is essentially unreliable, and useless if it relied upon observation or antibiotic administration as diagnostic criteria. Prospective studies must ensure that criteria for the appearance of the incision are explicit before the study starts, that all observers have been trained, and that inter-rater reliability is high.

Infection may occur within the surgical site at any depth, from the skin itself to the depths of the cavity that remains after resection of an organ. Superficial SSI involves tissues down to the fascia, whereas deep SSI extends beneath the fascia but not intracavitary. Infection of a visceral cavity (eg. Intra-abdominal abscess following intestinal surgery) is referred to as organ /

space infection).

Centers for Disease Control and Prevention (CDC) Definitions for Surgical Site Infection (SSI)

<u>Superficial incisional Surgical site infection</u>

Infection occurs within 30 days after the operation and infection involves only skin or subcutaneous tissue of the incision and

At least one of the following :

1. Purulent drainage, with or without laboratory confirmation, from the superficial incision
2. Organisms isolated from an aseptically obtained culture or fluid or tissue from the superficial incision.
3. Atleast one of the following signs or symptoms of infection: pain or tenderness, localized swelling, redness, or heat, and superficial incision is opened deliberately by the surgeon, unless the incision is culture-negative.
4. Diagnosis of superficial incisional surgical site infection by the surgeon

Deep incisional Surgical site infection : Infection occurs within 30 days

after the operation if no implant is in place, or within 1 year if implant is in place, and the infection appears to be related to the operation and Infection involves deep soft tissue (eg. Fascial and muscle layers) of the incision and

Atleast one of the following

1. Purulent drainage from the deep incision but not from the organ / space component of the surgical site.
2. A deep incision dehisces spontaneously or is opened deliberately by a surgeon when the patient has at least one of the following signs or symptoms: Fever (> 38C), localized pain, or tenderness, unless site is culture negative.
3. An abscess or other evidence of infection involving the deep incision is found on direct examination, during reoperation, or by histopathologic or radiologic examination.
4. Diagnosis of a deep incisional surgical site infection by the surgeon.

Organ / Space SSI

Infection occurs within 30 days after the operation if no impact is left in place or within 1 year if implant is in place and the infection appears to be related to the operation. And

Infection involves any part of the anatomy (eg. Organs or spaces) other than the incision, which was opened or manipulated during an operation. And

Atleast one of the following :

1. Purulent drainage from a drain that is placed through a stab wound into the organ / space
2. Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ / space
3. An abscess or other evidence of infection involving the organ / space that is found on direct examination, during reoperation, or by histopathologic or radiologic examination.
4. Diagnosis of an organ / space surgical site infection by the surgeon.

The degree of bacterial contamination of the surgical site has been well defined. Clean surgical procedures are those where the operation has affected only integumentary and musculoskeletal soft tissues. Clean-contaminated procedures are those where a hollow viscus (eg. Alimentary, biliary, genitourinary, respiratory tract) has been extensive introduction of bacteria

into a normally sterile body cavity, but for a period of time too brief to allow infection to become established during surgery (eg. Penetrating abdominal trauma, enterotomy during adhesiolysis for mechanical bowel obstruction). Dirty procedures are those where the surgery is performed to control established infection (eg. Colon resection for complicated diverticulitis).

Cellulitis refers to infection related erythema of skin (although other tissues may be affected) without drainage or fluctuance. Abscess refers to localized collections of purulent material within tissue. Necrotizing soft tissue infections (NSTI) invade tissue necrosis. When fascial is involved the infection is referred to correctly as necrotizing fascitis ; myonecrosis refers to involvement of underlying muscle. Necrotizing soft tissue infections are usually community acquired infections that require aggressive surgical debridement in addition to antibiotic therapy, and further discussion herein is not germane. Most SSIs do not cause extensive tissue necrosis, especially if the gastrointestinal tract has not been entered during surgery. Rare but dangerous exceptions to that rule are SSIs caused by Streptococcus pneumoniae Clostridium perfringens.

EPIDEMIOLOGY AND RISK FACTORS FOR SURGICAL SITE INFECTION

Numerous factors determine whether a patient will develop a SSI including factors contributed by the patient, the environment and the treatment. According to the National Nasocomal infection surveillance (NNIS) classification, the risk of surgical site infection increases with an increasing number of risk factors present, irrespective of the contamination of the incision and almost without regard for the type of operation.

Laproscopic surgery is associated with a decreased incidence of SSI under certain circumstances, which has required a modification of the National Nasocomal infection surveillance risk classification. For laparoscopic biliary, gastric, and colon surgery, one risk factor is subtracted if the operation is performed via the laparoscope – a new category has been created specifically for the circumstance, representing, essentially, - 1 risk factor. The reasons that laparoscopic surgery decreases the risk of SSI are potentially several, including decreased wound size, limited use of cautery in the abdominal wall, or a diminished stress response to tissue injury.

Laparoscopic appendectomy, on the other hand, is a unique circumstance. When no risk factors are present, the incidence of surgical site infection after laparoscopic appendectomy is reduced significantly, but if any risk factors is present (as would be the case with either perforated appendicitis or a procedure that last longer than 1h), then the advantage is lost.

More than 70% of surgical procedures are now performed on an outpatient basis, which poses major problems for surveillance of SSI. Although many surgical site infections will develop in the 5-10 days after surgery, a SSI will develop as long as 30 days after surgery and be attributable directly to the operation, long after even most inpatients have been discharged from the hospital. Estimates of the incidence of surgical site infections are thus dependent upon voluntary self reporting by surgeons, which may not occur for any of several reasons. Therefore, estimates of the incidence of surgical site infection in National Nasocomal infection surveillance are almost assuredly underestimates, although the data are the best that are available.

Host-derived factors may contribute importantly to the risk of SSI. Factors of importance include increased age, obesity, malnutrition, diabetes

mellitus, hypocholesterolemia and numerous other factors that are not accounted for specifically by the National Nasocomal infection surveillance (NNIS) system.

In one study of 2345 patients undergoing cardiac surgery, the overall incidence of SSI was 8.5% (199/2345). The relative risk for the development of SSI among diabetic patients was 2.29 (95% CI 1.15-4.54), and the relative risk among obese patients (body mass index > 30) was 1.78 (1.24-2.55). Malone et al, studied 5,031 patients who underwent noncardiac surgery at a Veterans Affairs hospital over a six year period ending in 1990. The overall incidence of SSI was 3.2% and independent risk factors for the development of infection included ascites, diabetes mellitus, postoperative anemia, and recent weight loss, but not chronic obstructive pulmonary disease, tobacco use or corticosteroid use. In a prospective study of 9,016 patients, 12.5% of patients developed a postoperative infection of some type within 28 days after surgery. The likelihood of readmission for infection management and of death were both 2.5% within the period. Multivariate analysis revealed that decreased serum albumin concentration, increased age, tracheostomy, and amputations were associated with an increased probability of an early

infection, whereas factors associated with readmission due to infection included dialysis shunt, vascular repair, and an early infection. Factors associated with 28 day mortality included increased age, low serum albumin concentration, increased serum creatinine concentration, and an early infection.

RISK FACTORS FOR THE DEVELOPMENT OF SURGICAL SITE INFECTION

1. Environmental factors

Contaminated medications

Inadequate disinfection / sterilization

Inadequate skin antisepsis

Inadequate ventilation

2. Patient Factors

Ascites

Chronic inflammation

Corticosteroid therapy (controversial)

Obesity

Diabetes

Extremes of age

Hypocholesterolemia

Hypoxemia

Peripheral vascular disease (especially for lower extremely surgery)

Postoperative anemia

Prior site irradiation

Recent operation

Remote infection

Skin carriage of staphylococci

Skin disease in the area of infection (eg. Psoriasis)

Under-nutrition

Treatment factors

Drains

Emergency procedure

Hypothermia

Inadequate antibiotic prophylaxis

Oxygenation

Prolonged preoperative hospitalization

Prolonged operative time.

MICROBIOLOGY

Inoculation of the surgical site occurs during surgery, either inward from the skin or out-ward from the internal organ being operated on, hence the rationale for skin preparation and bowel preparation with antiseptics or antibiotics, and prophylactic oral or parenteral administration of antibiotic prophylaxis. The microbiology of surgical site infection depends on the type of operation being performed, with an increased likelihood of infection caused by gram-negative bacilli after gastrointestinal surgery. However, most SSI are caused by gram positive cocci, including Staphylococcus aureus, coagulase-negative staphylococci (usually staphylococcus epidermidis), and Enterococcus sp., organisms that for the most part are skin-derived. With surgery of the head and neck (when pharyngoesophageal structures are entered) or intestinal surgery, enteric aerobic (eg. Escherichia coli) and anaerobic (eg. Bacteroides fragilis) bacteria may cause SSI. Perioperative antibiotic prophylaxis decreases only the incidence of surgical site infection, not that of other nosocomial infections. Therefore, for

prophylaxis of most procedures where prophylaxis is indicated, an agent with activity against community acquired (ie. methicillin sensitive) Staphylococcus aureus is appropriate.

Microbiology of Surgical Site infection :

Pathogen	Prevalence (%of isolates)
Staphylococcus	19
Coagulase-negative staphyloccus	14
Enterococcus sp.	12
Escherichia coli	8
Pseudomonas aeruginosa	8
Miscellaneous aerobic gram negative bacilli	7
Enterobacter sp	6
Streptococcus sp	6
Klebsiella sp	4
Miscellaneous anaerobic bacteria	3
Miscellaneous aerobic gram positive bacteria	2

ROLE OF PREOPERATIVE PREPARATION

Patient – Related :

Antiseptic baths preoperative bath / showers reduce the microbial load of the skin. For this purpose chlorhexidine has been found to be more useful

than povidine – iodine or triclocarbon medicated soap as it achieves a nine fold reduction in microbial load as compared to latter which achieve a reduction of 1 to 2 fold. In our country, where poor hygienic standards prevail, this practice of antiseptic baths preoperatively needs to be encouraged.

Preoperative hair removal :

Shaving the surgical site the night before an operation results in a significant risk for SSI microscopic cuts serve as foci for bacterial multiplication. Clipping hair immediately before an operation has been associated with decreased rates of surgical site infections and hence is the recommended practice. Shaving should be discouraged.

Patient skin preparation in the operating room :

The iodophors (povidine iodine 10%), products and chlorhexidine gluconate 4 percent in alcohol are the most commonly used agents. Alcohol has germicidal activity against bacterial, viruses and fungi, but spores can be resistant. One major deterrent to the use of alcohol in the operating room is its inflammability. Chlorhexidine gluconate and iodophores have broad

spectra of antimicrobial activity, but the former has greater residual activity and is not activated by blood or serum proteins. Prior to skin preparation, the skin should be freed of gross contamination. Applying antiseptic in concentric circles, beginning in the are of the proposed incision. The prepared area should be large enough to extend the incision or create new incision or drain sites, if necessary.

The Operating Room environment :

Much of what is taken for granted in the modern operating room can, if lapses occur, result in increased rates of SSI. The elements of proper operating room design, management, and comportment have been reviewed in an evidence – based manner.

Although such factors as proper sterilization technique and ventilation should not be the everyday concern of the surgeon, operating room personnel must remain vigilant. The surgeon must be responsible for his or her personal hygiene (eg. Hand scrubbing, hair) and that of the team of assistants. Recent data indicate that a brief rinse with soap and water followed by use of an alcohol gel hand rub is equivalent to the prolonged (and ritualized) session at the scrub sink.

Careful preparation of the skin with an appropriate antiseptic is essential. There are no data to show that one method (eg. Alcohol based vs povidone-iodine) is superior to an other, and also no data to raise concerns about lack of efficacy of the new quick drying gel formulations. However there are also no data to show that untreated or iodine impregnated adhesive plastic drapes reduce the risk of surgical site infection, so routine usage of such products may be foregone.

About 20% of surgical gloves fail during an operation, so there exists the possibility of contamination of the surgical field (as well as contact between surgeon and the patient's body fluids) ; therefore, attention must be paid to regular inspection of gloves during a procedure. Likewise, most surgical gowns in use offer limited protection (1.5 – 2 h at most) against strike through of fluids. It may be prudent to change gowns and gloves regularly (every 2h or so) during long procedures, and certainly if there is any evidence of loss if integrity of barrier materials.

Although most flora that pose a risk of infectivity in SSI are skin derived and inoculated during the procedure, airborne bacteria, especially

staphylococci, do pose some risk. Surgeons who are chronic nasal carriers of S.ureus have higher rates of SSI than do their non colonized brethren. It is recommended that surgical masks should cover the mouth and nose at all times, and that unnecessary conversation at the operating table be kept to a minimum.

Patients may become hypothermic during surgery if they are not warmed actively, owing to evaporative water losses, administration of room temperature fluids, and other factors. There is no longer a question whether maintenance of normal core body temperature is important for decreasing the incidence of surgical site infection ; the answer is unequivocally yes. Following the seminal observation that mild intraoperative hypothermia is associated with an increased rate of SSI following elective colon surgery, two other studies have provided corroboration. In one randomized study, 30 min of active preoperative warming reduced the rate of surgical site infection following minor clean operations.

ROLE OF PREOPERATIVE ANTIBIOTICS

What is decisive period :

The acute inflammatory, humoral and cellular process takes 4 hrs to mobilise body's response to a breach in its defense. This is called decisive period. And it is the time when the invading bacteria have a heard start in setting established in the tissues. So this is the optimal period of giving preoperative antibiotic.

The administration of antibiotics prior to surgery is common place and of proven benefit in many circumstances for the minimization of post operative surgical site infection. However, it is only the surgical incision that is afforded protection and antibiotics are not a panacea. If not administered properly, antibiotic prophylaxis will not be effective and may be harmful.

Some patients benefit from antibiotic prophylaxis whereas others may not. An increased risk of surgical site infection occurs with an increasing degree of wound contamination (eg. Clean Vs contaminated) regardless of other risk factors and as the number of risk factors increases for a given type of operation.

Antibiotic prophylaxis of clean surgery is controversial. Where bone is incised (eg. Craniotomy, sternotomy) or a prosthesis is inserted, antibiotic prophylaxis is generally indicated. The controversy exists in the case of clear surgery of soft tissues (eg. Breast hernia). A randomized prospective trial has shown some benefit of prophylaxis, but the results are confounded by higher than expected infection rates in the control group.) regardless of other risk factors and as the number of risk factors increases for a given type of operation.

Which antibiotic should be chosen from the plethora of available agents? Four principles should guide selection : The agent should be safe; the agent should have an appropriately narrow spectrum for coverage of relevant pathogens ; the agent should not be one that has a heavy reliance for clinical treatment of infection, owing to the possibility that resistance may develop if the agent is overused ; and the agent must be administered for a defined, brief period of time (certainly no more than 2 hrs). Most surgical site infection s are caused by gram positive cocci. The most common etiologic agent causing SSI after clean surgery is staphylococcus aureus, followed by S. epidermidis. Enterococcus aecalis, Escherichia coli, and Bacterioides fragilis

are common pathogens in surgical site infection after clean-contaminated surgery. The antibiotic chosen should be directed primarily against staphylococci for clean cases and high risk clean contaminated elective surgery of the biliary and upper gastrointestinal tracts. A first generation cephalosporin is the preferred agent for most patient, with a history of anaphylaxis to penicillin. Although methicillin resistant S. aureus Methicillin Resistant S. aureus (MRSA) has been isolated in the community from never hospitalized patients, vancomycin prophylaxis is appropriate only in institutions where the incidence of Methicillin Resistant S. aureus (MRSA) is high.

When should parenteral antibiotics be given for optimum effect? It is firmly established that the optimal time to give cephalosporin prophylaxis is within 2 hr prior to the time the incision is made. Antibiotics given sooner (except possibly for longer half life agents such as quinolones and metronidazole) are not effective, nor are agents that are given after the incision is closed. Antibiotics with short half lives should be re-dosed during surgery if the operation is prolonged or bloody and there is still some benefit if the initial antibiotic dose is given intraoperatively.

A simple way to organize antimicrobial prophylaxis (AMP) indications is based on using the surgical wound classification scheme as shown in table, which employs descriptive case features to post operatively grade the degree of intraoperative microbial contamination. A surgeon makes the decision to use antimicrobial prophylaxis (AMP) by anticipating preoperatively the surgical wound class for a given operation.

Surgical wound classification

Class I / Clean :

An uninfected operative wound with no inflammation and the respiratory, alimentary, genital or uninfected urinary tract not entered, in addition, clean wounds are primarily closed and if necessary, drained with closed drainage. Operative incisional wounds that follow non-penetrating (blunt) traumas should be included in the category if they meet the criteria.

Class II / Clean-Contaminated :

An operative wound where respiratory, alimentary, genital, or urinary

tracts are entered under controlled conditions and without unusual contamination. Specially, operations involving the biliary tract, appendix, vagina, and oroharynx are included in this category, provided no evidence of infection or major break in technique is encountered.

Class III / contaminated :

Open, fresh, accidental wounds, in addition, operation with major breaks in sterile technique (eg. Open cardiac massage) or gross spillage from the gastrointestinal tract and incisions in which acute, non-purulent inflammation is encountered are included in this category.

Class IV / Dirty – infected :

Old traumatic wounds with retained devitailized tissue and evidence of clinical infection or perforated viscera. This identification suggests the organisms causing post operative infection were present in the operative field before the operation.

ROLE OF POST OPERATIVE CARE

Blood transfusion

In surgery and trauma, blood transfusions are given commonly and may be life-saving; alternatives to transfusions in the acute setting are few, but for hemodynamically stable postoperative patients, hemoglobin concentrations of >7 g/ dL are well tolerated. Erythropoietin administration may decrease transfusion requirements of the" chronically critically ill" patient. An expanding body of evidence suggests that blood transfusion should be avoided, if possible.

Observations that blood transfusions are associated with increased rates of nosocomial infection are numerous. Blood transfusions have been associated with an increased risk of infection following penetrating abdominal trauma independent of related factors such as shock or acute blood loss and have been, related to increasing injury severity and increasing transfusion volume in unselected trauma patients. Data suggest that blood transfusion therapy of 6-20 units in the 'first 12 h following multiple trauma is associated with an increased risk of nosocomial infection The risk of

infection increased as the total transfusion volume increased, especially when units were transfused after more than 14 days of storage.

The postulated "storage lesion" is complex, but includes changes in oxygen affinity, red blood cell deformability, shortened circulation time, and the biologic consequences of cytokine generation and release. Recently, observational studies have suggested that transfusion of critically ill patients increases the risk of nosocomial infection may worsen Multi organ disease syndrome (MODS), and increases mortality.

Hyperglycemia, nutrition, and controlof blood sugar

Hyperglycemia has several deleterious effects upon host immune function, most notably impaired function of neutrophils and mononuclear phagocytes. It is possible also that hyperglycemia is a marker of the catabolism and insulin resistance associated with the surgical stress response, and that exogenous insulin administration may ameliorate the catabolic state.

Increasing evidence indicates that poor control of blood glucose during surgery and in the perioperative period increases the risk of infection, and worsens outcome from sepsis. Diabetic patients have a higher risk of infection of both the sternal incision arid the vein harvest incisions on the

lower extremities. Tight control of blood glucose by the anesthesiologist during surgery must be accomplished to decrease the risk, and that control must extend into the immediate postoperative period as well. Moderate hyperglycemia (>200 mg/ dL) at any time on the first postoperative day increases the risk of surgical site infection after non-cardiac surgery Hyperglycemia during the immediate phase of trauma resuscitation increases the risk of nosocomial infection and death after trauma. Exogenous insulin administration to keep blood glucose concentrations of <120 mg/ dL was associated with a 40% decrease of mortality among critically ill surgical patients with sepsis. The need to manage carbohydrate metabolism carefully has important implications for the nutritional management of surgical patients.

Gastrointestinal surgery may render the gastrointestinal tract unusable as a route of feeding, sometimes for prolonged periods. Ileus is common in surgical Intensive care units (ICUs), whether from traumatic brain injury, narcotic analgesia, prolonged bed rest, inflammation in proximity to the peritoneal envelope (lower lobe pneumonia, retroperitoneal hematoma, fractures of the thoraco-Iumbar spine, pelvis, or hip), or other causes.

Parenteral nutrition is relied upon for feeding, despite evidence of a lack of efficacy and the possibility of hepatic dysfunction; hyperglycemia may be an important complication as well. Every effort should be made to provide enteral feedings, including the use of pro-motility agents such as erythromycin. Substantial evidence indicates that enteral feedings reduce the risk of nosocomial infection by nearly one half among critically ill and injured patients.

CELLULAR, BIOCHEMICAL, CLINICAL & PATHOLOGICAL ASPECTS OF WOUND HEALING

Healing by first intention :

The incision causes death of limited number of epithelial and connective tissues as well as distrubtion of epithelial basement membrane continuity. The narrow incisional space immediately fills with collotted blood contains firbrin and blood cells dehydration of the surface clot forms the well known scap that covers the wound. The healthy process follow services of sequential steps.

Within 24 hrs :

Neutrophic apex at the margins of the incision. Many towards the fibrin clot. In 24 to 48 hrs spur of epithelial cells moves from wound edges along the cut margins of the dermis depositing basement membrane components as they move. They fuse in the midline beneath the surface scab producing consunience but thin epithelial layer that closes the wound.

By day 3 :

The neurophils have been longer replaced by macrophages. Granulation tissue progressively invades the incision space. Collagen fibres are point in the margin of the incision but at first they are vertically oriented and don't bridge the incision. Epithelial cell proliferation thickens the epidermal layer.

By day 5 :

The incisional space is filled with granulation tissue. Neovascularisation is maximal. Collagen fibrils become more abundant and begin to bridge the incision. The epidermis recovers its normal thickeness and differentiation of surface cells yields a mature epidermal architecture with surface keratinization.

2nd week :

There is continued accumulation of collagen and proliferation of fibroblasts. The leukocytic infiltration, odema and increased vascularity have largely disappeared. At this time the long process of bleaching begins accomplished by the increased accumulates of collagen with in the incisional scar accomplished by regression of vascular channels.

By the end of 1st month :

Scar is mode of a cellular connective tissue decend of inflammatory

infiltration could now try intact epidermis. The dermal appendages that a destroyed at the line of incision are permanently lost. Tensile strength of the wound increase thereafter, but it may take months for the wounded area to obtain its maximal strength.

Healing try 2nd intention (wound with separated edges)

When there is more loss of cell and tissue as in surface wounds that create large defects the reparative process is more complicated. Regeneration of parenchymal cells causing completely restore the original architecture and less abundant granulation tissue grows in from the margin to complete the repair. This form of healing is referred to as secondary union or healing by second intention secondary healing differs from primary in several respects.

Inevitably large tissue defect generation a large fibrin clot that fills the defect and more necrotic debris and caudatic that must be removed. Consequently the inflammatory reaction is more intensive.

Much large amount of granulation tissue are found perhaps the feature that most clearly differentiate in the wound contracture which occur in large surface wounds. Initial step of wound contracture is formation of network of action containing fibroblasts at the edge of the wound. Permanent wound

contractive requires action of myofibroblasts altered fibroblasts that have the ultra structural character of smooth muscle cells. Contraction of there cells at the wound edge decreases the gap between the dermal edges of the wound. Substantial scar formation and thinning of the epidermis.

Mechanisms of Wound Healing :

Wound healing, as we have seen, is a complex (but orderly) phenomenon involving a number of processes, including induction of an acute inflammatory process by the wounding, regeneration of parenchymal cells, migration and proliferation of both parenchymal and connective tissue cells, synthesis of Extra cellular matrix (ECM) proteins, remodeling of connective tissue and parenchymal components, and collagenization and acquisition of wound strength. As noted the deposition of connective tissue matrix, particularly collagen its remodeling into a scar and the acquisition of wound strength are the ultimate effects of orderly wound repair. We shall end this discussion of wound healing by considering the determinants of wound collagenization.

Collagen synthesis : Degradation and wound strength :

Collagen is the most common protein in the animal world, providing the

extracellular frame work for all multicellular organisms. Without collagen, a human being would be reduced to a clump of cells, interconnected by a few neurons. The essential product of the fibroblast, collagen, ultimately provides the tensile strength of healing wounds. On the basis of the biochemical composition of the chains that make up the triple helix of the collagen molecule, some 14 types of collagen can be discerned, of which the most well characterized. Types I, II and III are the interstitial or fibrillar collagens. Types IV, V and VI are amorphous and are present in interstitial tissue and basement membranes.

Following synthesis on ribosomes, the alpha chains are subjected to a number of enzymatic modifications, including hydroxylation of praline in the alpha position, providing collagen with its characteristic high content of hydroxyproline (about 10%). This hydroxylation, which is dependent on the availability of ascorbic acid (vitamin C) is important because it is necessary to hold the three alpha chains together.

At this stage the precollagen molecule is still soluble and contains extra length of polypeptide and C terminal at the ends of the chain. During or shortly after excretion from the cell, procollagen peptidases clip the terminal

peptide chains, promoting formation of fibrils. True fibrils form in the extracellular space, and these collagen fibrils give strength to connective tissue. A critical extracellular modification is lysyl hydroxylysyl oxidation, because this results in cross linkages between alpha chains of adjacent molecules and is the basis of the structural stability of collagen. Cross linking is a major contributor to the tensile strength of collagen.

As previously described, collagen synthesis by fibroblasts begins early in wound healing, by day 3 or 5, and continues for several weeks, depending on wound size. Collagen synthesis is stimulated by several factors, including growth factors (PDGF, FGF, TGF-beta) and cytokines (IL-1, IL-4), which are secreted by leukocytes and fibroblasts in healing wounds. Net collagen accumulation, however depends not only on synthesis but also on collagen degradation.

Degradation of collagen and other Extra cellular matrix (ECM) proteins is achieved by a family of metalloproteinases, which are dependent on zinc ions for their activity. (Neutorphil elastase, cathepsin G, kinins, plasmin, and other important proteolytic enzymes mentioned earlier can also degrade extra cellular matrix components, but they are serine proteinaes, not metal

loenzymes). Metalloproteinases consist of interstitial collagenases, which cleave the fibrillar collagen types I, II and III ; gelatinases (or type IV collagenases) which degrade amorphous collagen as well as fibronectin and amorphous collagens. These enzymes are produced by several cell types (fibroblasts, macrophages, neutrophils, synovial cells and some epithelial cells) and their secretion is induced by many stimuli including growth factors (PDGF, FGF), cytokines (IL-1, TNF-alpha) and phagocytic stimuli. Collagenases cleave collagen under physiologic conditions, cutting the triple helix into two unequal fragments, which are then susceptible to digestion by other proteases. This is potentially harmful to the organism, but the enzyme is elaborated in a latent (procollagenase) form that can be activated by chemicals (HOCI produced, as you recall, during the oxidative burst of leukocytes) and proteases (plasmin). Once formed, activated collagenases can be rapidly inhibited by a family of specific tissue inhibitors of metalloproteinase (TIMP), which are produced by most mesenchymal cells. There are thus multiple checks against the uncontrolled action of these proteinases. Nevertheless, it is thought that the collgenases play a role in degrading collagen in inflammation and wound healing. Degradation aids in

the debridement of injured sites and also in the remodeling of connective tissue necessary to repair the defect. Indeed, collagenases and their inhibitors have been shown to be spatially and temporally regulated in healing burn wounds.

We now turn to the questions of how long it takes for a skin wound to achieve its maximal strength and which substances contribute to this strength. When sutures are removed, usually at the end of the first week, wound strength is approximately 10% of the strength of unwounded skin, but it increases rapidly over the next 4 weeks. This rate of increase then slows at approximately the third month after the original incision and then reaches a plateau at about 70 to 80% of the tensile strength of unwounded skin, which indeed may persist for life. The recovery of tensile strength results from increased collagen synthesis exceeding collagen degradation during the first 2 months and from structural modifications of collagen fibers (cross-linking, increased fiber size), when collagen synthesis ceases at later times.

We can conclude this discussion of wound healing and collagenization by emphasizing that the healing wound, as a prototype of many other forms of tissue repair, is a dynamic and changing process. The early phase is one

of inflammation, followed by a stage of fibroplasias, followed by tissue remodeling and scaring. Different mechanisms occurring at different times trigger the release of chemical signals that modulate the orderly migration, proliferation, and differentiation of cells and the synthesis and degradation of extra cellular matrix proteins. These proteins, in turn, directly affect cellular events and modulate cell responsiveness to soluble growth factors.

The magic behind the seemingly precise orchestration of these events under normal conditions remains beyond our grasp, but almost certainly lies in the regulation of specific soluble mediators and their receptors on particular cells, cell-matrix interactions, and a controlling effect of physical factors, including forces generated by changes in cell shape and plasticity.

Pathologic aspects of inflammation and repair

The usual manifestations of inflammation and repair and we reviewed the orderly healing of wounds in normal persons. But these processes are modified by a number of known influences and some unknown ones, frequently impairing the quality and adequacy of both inflammation and repair.

Many systemic and local host factors influence the adequacy of the

inflammatory reparative response. Nutrition has profound effects on wound healing. Protein deficiency, for example and particularly vitamin C deficiency, inhibit collagen synthesis and retard healing. Glucocorticoids have well documented anti-inflammatory effects that influence various components of inflammation and fibroplasias. Of the local factors, infection is the single most important cause of delay in healing. Mechanical factors such as increased abdominal pressure may cause rupture of abdominal wounds, called wound dehiscence. Inadequate blood supply usually caused by arteriosclerosis or venous abnormalities that retard venous drainage also impair healing. Finally, foreign bodies such as necessary sutures or fragments of steel, glasss or even bone constitute impediments to healing.

Aberrations of growth may occur even in what may begin initially as normal wound healing. The accumulation of excessive amounts of collagen may give rise to a raised tumorous scar known as a keloid. Keloid formation appears to be an individual predisposition, and for reasons unknown this aberration is somewhat more common in blacks. We still do not know the mechanisms of keloid formation. Another deviation in wound healing is the formation of excessive amounts of granulation tissue, which protrudes above

the level of the surrounding skin and in fact blocks re-epithelialization. This has been called exuberant granulation or, with more literary fervor, proud flesh. Excessive granulations must be removed by cautery or surgical excision to permit restoration of the continuity of the epithelium. Finally (fortunately rarely), incisional scars or traumatic injuries may be followed by exuberant proliferations of fibroblasts and other connective tissue elements that may, in fact, recur after excision. Called desmoids or aggressive fibromatoses, these lie in the interface between benign proliferations and malignant (though low-grade) tumors. Indeed, the line between the benign hyperplasias characteristic of repair and neoplasia is frequently finely drawn.

The mechanism underlying the fibroplasias of wound repair – cell proliferations, cell – cell interactions, cell-matrix interactions, and ECM deposition – are similar to those that occur in the chronic inflammatory fibrosis of such diseases as rheumatoid arthritis, lung fibrosis, and hepatic cirrhosis. Unlike orderly wound healing, however, the diseases are associated with persistence of initial stimuli for fibroplasias or the development of immune and autoimmune reactions in which lymphocyte-monocyte interactions sustain the synthesis and secretion of growth factors

and fibrogenic cytokines, proteolytic enzymes and other biologically active molecules. Collagen degradation by collagenases, for example, which is important in the normal remodeling of healing wounds, causes much of the joint destruction in rheumatoid arthritis.

OBSERVATION AND RESULTS

During the study period of 2 years, 200 cases were observed of which 100 elective cases and 100 emergency cases.

On 50 elective cases and emergency cases povidone iodine was used as skin antiseptic for various procedures. Likewise 4% Chlorhexidine gluconate was used as skin antiseptic on 50 elective cases and emergency cases for various procedures.

On observing the patient in the immediate post operative period in the aspect of surgical site infection we got the following results.

In patients to whom we used Povidone iodine as skin antiseptic the following rate of SSI observed.

In Elective Cases

The rate of Superficial Surgical site infection - 20%

Deep Surgical site infection - 4%

In emergency cases

The rate of Superficial Surgical site infection - 26%

Deep Surgical site infection - 8%

In patients on whom we used 4% Chlorhexidine gluconate as skin antiseptic the following rate of SSI observed.

The rate of Superficial Surgical site infection - 10%

Deep Surgical site infection - 2 %

Emergency cases :

The rate of Superficial Surgical site infection - 16%

Deep Surgical site infection - 4%

From the above observation it shows that in case of elective procedures there is 50% reduction of superficial Surgical site infection and 50% reduction of deep Surgical site infection and also in case of emergency procedures there was a 39% reduction of superficial Surgical site infection and 50% reduction of Deep Surgical site infection.

DISCUSSION

Previous studies showed that Chlorhexidine gluconate is effective in reducing bacterial counts on the skin. Kaiser et al., found that Chlorhexidine gluconate was more effective than povidone-iodine in reducing skin colonization with staphylococci and that repeated applications of Chlorhexidine gluconate were superior to a single applications. A fullbody 4 percent Chlorhexidine gluconate shower reduces bacterial counts at abdominal and inguinal sites, and a 4 percent Chlorhexidine gluconate two minute surgical scrub has been shown to reduce microbial counts better than povidone-iodine.

Recent data from DeBaun confirms a 99.9 percent reduction in MRSA after three minutes of exposure to Chlorhexidine gluconate and Acinetobacter after 15 seconds of exposure. These studies show the effectiveness of Chlorhexidine gluconate in reducing bacterial counts, which may, in turn, reduce surgical site infection rates. Given today's healthcare environment and patient concerns with Methicillin resistant S.aureus (MRSA), which they refer to as 'superbugs', it seems prudent to have patients apply full-skin

antisepsis before entering the operating room.

Although it would be expected that skin antisepsis would reduce SSI rates, studies of previously available skin antiseptics yielded mixed results.

Chlorhexidine gluconate solution reduced surgical site infections compared with iodine-based products in one study, whereas four studies of iodine-based preps found no reduction in Surgical site infections. These studies examined the benefits of using a 4 percent Chlorhexidine gluconate solution.

Edmiston et al, found that although both 4 percent Chlorhexidine gluconate solution and a 2 percent Chlorhexidine gluconate impregnated cloth were effective at reducing bacterial counts.

During the study period of 2 years on patients in the aspect of SSI with the use of above mentioned methods strictly followed. In case of superficial SSI in case of elective procedures there is 50% reduction and in case of Deep surgical site infection 50% reduction observed in elective cases and 39% in superficial surgical site infection and 50% Deep surgical site infection reduction in emergency cases.

The strength of this study were its prospective nature and that a large number of procedures were monitored. Because this was a pilot study and not a controlled study, further analysis of results and direct comparison with another product was not done. Also the lack of a control group prevented estimation of the relative benefits of avoiding hypothermia in the study group.

CONCLUSION

Surgical site infections are a serious medical problem associated with increased morbidity and mortality and increased medical care costs. This study showed that a new presurgical preparation protocol using warmed 4% Chlorhexidine gluconate skin antiseptic of surgical site infection compared with a historical observation period. This reduction in surgical site infections, in turn, led to reduced medical costs, even when the cost of the Chlorhexidine gluconate was taken into account. Because of these promising results, our facility has chosen to continue to use the 4 percent Chlorhexidine gluconate antiseptic as skin antiseptic.

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PROFORMA

Name : Age :

Sex : Inpatient No. :

Unit :

D.O.A : D.O.S :

Procedure → Elective
Procedure → Emergency

Material → Chlorhexidine 4%
Material → Povidone Iodine 7.5%

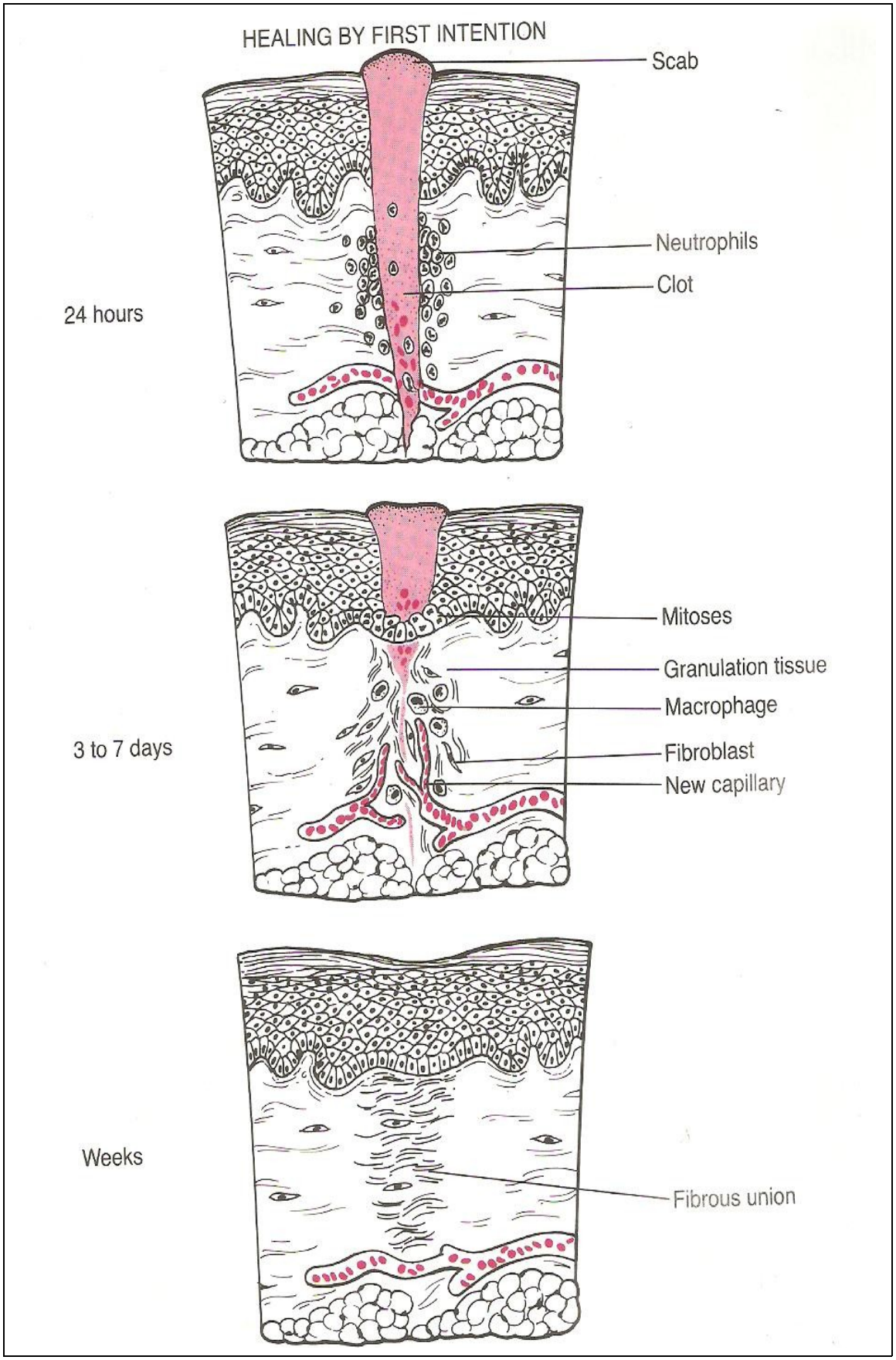
Method :

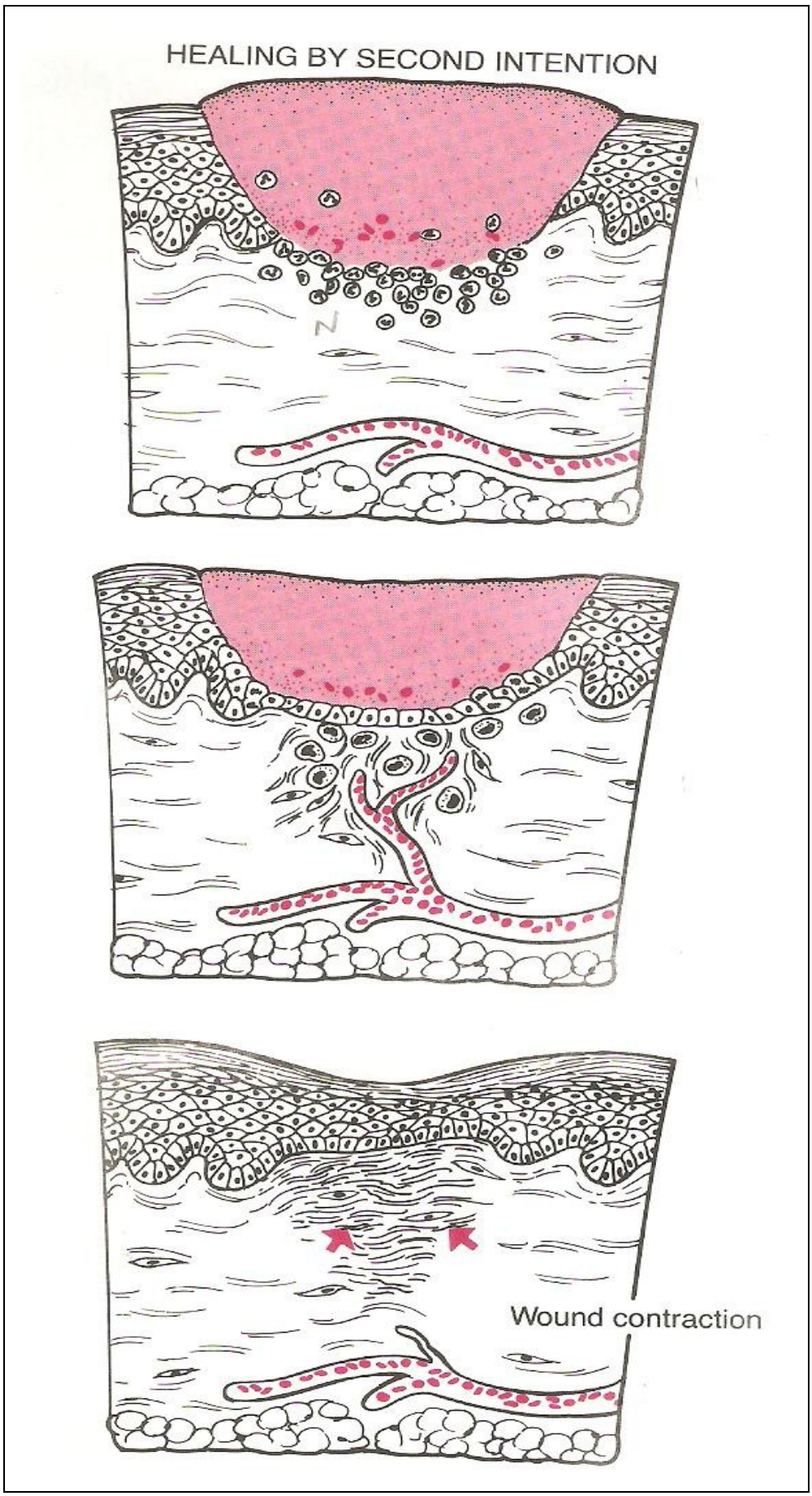
Microbiology result :

Follow up :

Outcome of the study :

Conclusion :


HEALING BY FIRST INTENTION
Scab
Neutrophils
Clot
24 hours
Mitoses
Granulation tissue
Macrophage
3 to 7 days
Fibroblast
New capillary
Weeks
Fibrous union


HEALING BY SECOND INTENTION
Wound contraction